believes that there is some defect with the present claim of priority. The Examiner has requested that Applicant cancel non-elected claims subjected to the restriction requirement. Applicant hereby cancels claims 1-59, 65, 73, and 74.

Response to Arguments and Amendments

Applicant thanks the Examiner for the indication that the rejection of claims 64 and 67 under 35 U.S.C. §112, second paragraph has been withdrawn.

The rejection of claims 60-64 and 66-72 under 35 U.S.C. §112, first paragraph has been maintained. The Examiner has indicated that the rejections have been maintained because no evidence was presented that "regulation of lysosomal pH has been demonstrated using any UCP inhibitors, nor has the prevention or treatment of infectious diseases been shown in an organism comprising the administration of any UCP inhibitors." (Paper No. 13, page 3, last paragraph).

In response to the rejection, Applicant hereby presents a Declaration by Dr. Karen Newell, the inventor of the above-identified patent application, demonstrating that 1) UCP inhibitors regulate lysosomal pH, and 2) UCP inhibitors result in the prevention or treatment of infectious diseases.

The data attached as Exhibits 1-3 demonstrate that UCP inhibitors regulate lysosomal pH and that such regulation is useful in treating infectious disease. In order to demonstrate the effect of UCP in the lysosome, UCP2 knock-out mice were studied. As shown in Exhibit 1, UCP2 knock out mice have more acidic lysosomes than mice with functional lysosomal UCP2. The data demonstrate that the inhibition of UCP2 in lysosomes of mice regulates pH levels in the lysosome. Exhibits 2-3 demonstrate that the same knock out mice have a physiological response which is consistent with increased resistance to infection. The data show that UCP2 knock out mice have higher levels of B cells (Exhibit 3) and express more MHC class II on the cell surface (Exhibit 2, IAb refers to MHC class II for this strain of mice, isotype controls were subtracted out). Additionally, the study by Arsenijevic *et al.*(*Nature Genetics*, 2000, 26(4):387-8; cited in an IDS dated May 15, 2001 and described in response to the last office action) has shown that control mice are highly susceptible to *Toxoplasma gondii*, but that UCP2 knock out mice become extremely resistant to this same organism when they lack a functional UCP2.

Thus, Applicant has demonstrated with *in vivo* data that 1) UCP inhibitors regulate lysosomal pH, and 2) the same UCP inhibitor cause physiological changes which are useful in the prevention or treatment of infectious diseases.

Summary

It is believed that the above evidence should be sufficient to overcome the rejection raised in the Office Action. It is respectfully requested that the rejection of the claims be withdrawn.

Respectfully submitted,

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Docket No. V00139.70059.US
Date: October 22, 2002